Mettler (L. H.)

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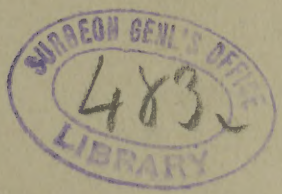

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SOME REMARKS ON PNEUMONIA AND THE CAUSE OF HEART FAILURE,

WITH REPORT OF A CASE.

By L. HARRISON METTLER, A. M., M. D.,
CHICAGO.

At present there are two views in regard to the cause of the much-dreaded heart failure of pneumonia. In a loose way they may be characterized as the *mechanical* and *chemical* theories. The mechanical theory is the older of the two, and attributes the cardiac weakness occurring about the crisis of the disease to the changes produced in the heart muscle by the prolonged high temperature and to the difficulty which the heart experiences in forcing the blood through the obstructed lungs. The chemical theory assigns the heart failure to a nervous source. It insists that the nervous apparatus of the circulatory system is poisoned by certain toxic matters, ptomaines and other substances, retained in the blood. It fails to explain just what the nature of the poison is, how it is generated, and in what particular manner it affects the vaso-motor nervous apparatus. Many writers approve of both the mechanical and chemical theories, and accord an equal importance to both as the causative agents in pneumonic heart failure.

In the absence of more positive knowledge, this may be a discreet position to assume, but in the majority of cases it seems to me that the chemical theory offers the more satisfactory explanation. My attention was forcibly turned to this subject by the following case, which is not reported, however, as an unusual one by any means:

H. J. B., a young man, twenty-two years of age, with a fairly good family history, was one day overcome during business hours with a sudden attack of vertigo and general prostration. He did not lose consciousness, but at once went home, took to his bed, and attempted to treat himself with some simple home remedies. When I first saw him, September 15, 1891, I learned the following facts: He had been considerably annoyed with nasal catarrh, and for the last few weeks had been indulging in frequent cold baths. He described a kind of "queer feeling" over his whole body, and complained of a severe aching in the neighborhood of his joints. Near the base of the left lung he felt a short, sharp pain whenever he took a long breath. His bowels had been averaging about two movements in the twenty-four hours. His tongue was thickly coated, red along the edges, and deeply cracked down the middle. He was made uncomfortable by a constant bad taste. Tea and coffee he indulged in freely, but no alcoholics; smoked on an average two cigars a day; and as a general thing enjoyed a good appetite. He was afraid that his heart was diseased; and between the difficult, shallow breathing and the dread of impending dissolution, it was apparent that he was in great distress. His face was hot and flushed. The pulse was 96, the temperature 103°. He complained especially of an intense throbbing headache in the frontal region. There was no epistaxis or other indications of typhoid poisoning. Examination of the chest revealed a slight dullness near the base of both lungs, more marked, however, on the left side. There were no râles or perceptible fremitus. In fact, the physical signs at this time were so insignificant as to be almost nil. I gave at once a tablet containing a quarter of a grain of sulphate of morphine, one one hundred and twentieth of a grain of sulphate of

atropine, and two drops of the tinctur of aconite root. He was then ordered to take at once eight grains of the sulphate of quinine, continuing afterward with two grains every two hours. A mustard poultice was to be applied to the chest, an abundant milk diet allowed, and a strong mustard foot-bath taken at bed-time.

September 16th.—The patient slept during the night, but was extremely restless in his sleep. The mustard was removed from the chest before the skin had become reddened, otherwise the directions were carried out explicitly. The morning pulse and temperature were 96 and 102°. The tongue was redder and more deeply serrated. Absolute rest in bed was enjoined, and the mustard reapplied to the chest. He was ordered to take two drops of tincture of aconite root with three drops of tincture of opium in water every hour, two grains of sulphate of quinine every two hours, milk punch, lemonade, and cracked ice *ad libitum*. In my afternoon visit I found the patient feeling somewhat easier, but still restless and complaining of the pain near the base of the left lung. The pulse registered 100, the temperature 102°. I then dry-cupped him very freely, both anteriorly and posteriorly, over the lungs, which he declared gave him almost instant relief. Eight grains of the sulphate of quinine were prescribed at once, and ten grains more ordered to be taken an hour later. The mustard was soon again applied to the chest, and after its removal the upper part of the patient's body was wrapped in a thick wadding of raw cotton covered with oiled silk. At bed-time he was ordered to begin taking the aconite and opium drops every three hours for the rest of the night, and occasionally a teaspoonful of whisky in sweetened water.

17th.—The night had been close and the patient suffered a good deal from restlessness. He had been able to take very little nourishment and almost no whisky, because the stomach refused to retain either. The bowels moved about six times, with loose but not particularly offensive stools. The intolerable pain about the lungs had completely disappeared, but there still remained considerable headache. The pulse was 86, the temperature 102°. I ordered ten grains of sulphate of quinine

at once, to be followed by two grains every two hours in conjunction with the aconite and opium drops. The chest was kept enveloped in cotton. A teaspoonful of brandy was administered every three hours, and such nourishment as milk, beef tea, broths, and plain jellies given every two hours. In the afternoon the pulse numbered 84 and the temperature registered 100·8°. The patient said the brandy "braced him up," and in every way he seemed much better. The two-grain quinine pills, with the aconite and opium drops, were continued every four hours, while some nourishment, with a teaspoonful of the brandy, was given every two hours.

18th.—Somewhat restless during the night, but the patient declared he felt very comfortable this morning, except for a kind of oppressive heaviness near the lower part of both lungs. Indeed, he felt so easy that he insisted upon having the barber come to shave him. At 6 A.M. the temperature had been at 100°; about the middle of the forenoon I found it to be 101° and the pulse at 76, full and strong. A slight headache only was complained of. The lungs seemed to be clearing up, and the heart showed no signs of weakness. I stopped the brandy as well as the aconite and opium drops. Nourishment was urged every two hours, and I ordered the application of a large flaxseed poultice on the lower part of the lungs, a Seidlitz powder, two grains of quinine every two hours, and a dessertspoonful of the following combination every three hours:

℞ Ammon. carb. ..... gr. lxxx;
Pulv. acac. et sacchar ..... āā q. s.;
Sp. lavandul. comp. ..... ℥ ij;
Aquæ ..... ad ℥ iv. M.

19th.—The patient had four quite loose stools since the day before. There was no pain about the chest and scarcely any headache, but he complained somewhat of the fullness of the lungs and the difficulty he had in breathing. The dullness on percussion was still quite marked about the base of the left lung; somewhat less so over the same area of the right lung. I again cupped the chest freely, and ordered the flaxseed poultices to be changed every fifteen minutes. The quinine pills and ammonium-carbonate mixture were continued, and the fol-

lowing suppository used at 10 A. M., 4 and 8 P. M., and 6 A. M. the next morning:

℞ Pulv. opii..... gr. ss.;
Quinin. sulph..... gr. x;
Ol. theobrom..... q. s. M.

20th.—The patient was much easier than the day before. The pulse and temperature registered 78 and 100·5°. The pulmonic dullness was still marked, while expectoration streaked with slight rusty sputum was beginning to be freer. The dullness at the base of the right lung seemed to be increasing, while that of the left side was diminishing. I continued the use of the poultices and suppository, and ordered the following mixtures to take the place of the quinine pills and ammonium carbonate:

℞ Potas. iodid..... ℥ ij;
Ammon. chlorid..... ℥ jss.;
Mist. glycyrrhiz. comp..... ℥ vj.
M. Sig.: Tablespoonful four times a day.

℞ Pulv. digitalis..... gr. ss.;
Quinin. sulph..... gr. j;
Ext. opii..... gr. ss.;
Ext. ipecac..... gr. ¼.
M. et ft. in pil. no. j.
Sig.: One three times a day.

A small quantity of stimulus was allowed when there was marked weakness.

21st.—The patient expectorates freely, there is no more blood in the sputum, the pulse is full and regular, and the temperature marks 99·5°. Breathing is easier, and, in his own words, he "feels fifty per cent. better than ever." Same treatment continued.

22d.—Both pulse and temperature were normal this morning. Expectoration was free and appetite rapidly returning. The only change made in the treatment was the omission of the suppositories. So well, apparently, was the patient that, although I recommended quiet and rest in bed for a few days longer, I said I would not call as frequently, but left word that they should send for me immediately if any unusual symptoms

made their appearance. I was in doubtful anticipation of the crisis.

About 5.30 P. M. I was hurriedly summoned and found that the patient had experienced, about an hour previous, a violent chill and a sudden feeling of impending death. His features showed alarm, although he was somewhat stupid and indifferent. He was covered with a cold, clammy sweat. The respiration was slow, regular, and labored. The pulse-beat was 58 and full, while the temperature continued to be normal. The lips were pale, the extremities cold, and the eyes dark and sunken. I recognized the condition as critical heart failure. The lungs upon examination were found to be comparatively clear. The heart was hypertrophied, probably as the result of the three or four previous attacks of pneumonia which the mother now told me he had passed through, but in none of which he had experienced the sudden prostration of this attack. In consultation, Dr. H. H. Deming confirmed both my opinion and treatment of the case. I at once stopped all medicine save the ammonium carbonate mixture, and increased the amount of alcoholic stimulants. At 9 P. M. there was another violent chill, longer than the first, more prostrating, and followed by the same cold sweat, which quite wet the bed-clothing. The accompanying stupor was also the same as that following the previous chill. The anxiety and nervous depression were extreme. The pulse was weak, thready, and beat 50 to the minute. Hot bottles were placed in the bed and half an ounce of whisky in water given every hour. The ammonium-carbonate mixture which had been ordered on September 18th was resumed, and administered every three hours, together with the following pill every six hours:

℞ Pulv. digitalis..... gr. ss.;
Quinin. sulph..... gr. j;
Ext. gentian..... q. s. M.

Remaining with the patient myself the greater part of the night, I devoted all my attention to stimulating and sustaining the heart's action.

23d.—Called at 7 A. M. and found much less nervous depression, though an extreme degree of physical weakness. There

was no cough. The respiration was slow and labored, the pulse 50, and the temperature 97·8°. The medicinal treatment was continued with free stimulation. Nourishment in small amounts was allowed every hour, consisting of milk with lime water, beef tea, mutton broth, milk toast, jellies, etc.

24th.—At 8 A. M. the condition of the patient exhibited a marked improvement. He was brighter and stronger. The pulse varied between 55 and 60. The surface of the body was warmer and there were no more chills, though he continued to perspire abundantly. In place of the digitalis pill which he had been taking, I ordered the night before the following prescription, to which I attribute the decided change for the better this morning:

℞ Strychnin. sulph..... gr. $\frac{1}{80}$;
Tinct. digitalis..... gtt. v;
Tinct. gentian comp..... ad ʒj.
M. Sig.: To be taken every six hours.

The nourishment consisted of milk, beef tea, broths, toast, calves'-foot jelly, and Parke, Davis, & Co.'s hæmoglobin compound in teaspoonful-and-a-half doses three times a day.

25th.—The condition about the same. Every two hours some nourishment with an ounce of whisky was given. The ammonium-carbonate mixture was continued every two hours and the strychnine administered every four hours.

26th.—The patient passed a comfortable night and slept well. He took about a quart of milk, a full cup of beef tea, and the hæmoglobin compound. About 3.30 P. M. to-day there was another chill, not so severe as the previous ones, but followed by the same profuse perspiration. The temperature registered 97° and the pulse 52. There was no difficulty of breathing, no cough, but excessive prostration. I ordered the whisky in half-ounce doses every hour, the ammonia mixture every two hours, the strychnine every four hours, and an addition to the diet of some cup custard with a soft-boiled egg the following morning.

27th.—The patient slept soundly, but awoke feeling chilly and bathed in a profuse perspiration. His general condition remained about the same. The temperature was 98°, the pulse

52. The appetite was reviving and he was allowed to-day a small piece of mutton chop and some milk toast. The same medicinal treatment was continued.

From this time on there was nothing of special moment either in the character of the case or its treatment. Occasionally there would be a slight chill followed by a profuse sweat. The pulse continued to remain at 52, and the temperature fluctuated between 97° and 98°. The diet was closely watched and cautiously increased. The stimulants and medicines pushed, changed, or withdrawn as the conditions seemed to require.

October 2d.—The pulse was still at 52 and the temperature 97°. Patient ate some ice-cream upon his own responsibility and had a slight attack of colic. He now took three regular meals a day, an ounce of whisky every two hours, the strychnine every four hours, and a teaspoonful of the following combination after each meal:

℞ Pepsin in lamel. (P. D. & Co.)..... ʒj;
Acid. hydroch. dil ..... ℥ss.;
Syrup. limon..... ʒij;
M. Aquæ ..... ad ℥iij.

3d.—The pulse steadily rose to 68 to-day. Stimulants were gradually withdrawn. General tonics with cod-liver oil and maltine were prescribed. A day or two later the patient began getting out of bed, and on October 10th he called at my office perfectly well but very weak.

This case, which, as I have said, is not presented on account of any unusual features, affords several lessons, and therefore I report it as a text for the following remarks: I have long been convinced that croupous pneumonia is a systemic disease with a local manifestation in the lungs. It attacks the individual suddenly and its first effects are felt throughout the whole constitution. Its nervous manifestations especially ally it to the other specific constitutional affections. It is certainly not a simple inflammation, as proved by very many of its characteristics, and especially by the re-

sults obtained when a line of treatment is adopted in accordance with the idea of its being an inflammation. Were it an inflammatory disease there would have been formulated long ago a method of treatment more universally adopted than any now employed. The late discussions and comparisons among the physicians of Paris in regard to the treatment of pneumonia revealed a most lamentable uncertainty of knowledge. Each reporter vaunted his own method, and scarcely two were found to exhibit any sort of agreement. As has been pointed out by various experimenters, the inhalation of hot and cold vapors and noxious gases, the injection of caustic ammonia and mercury, as well as traumatic injuries, all produce catarrhal but never croupous pneumonia. The bacterial origin of the latter has the support of the strongest arguments, but the nature and influence of the micro-organisms are yet far from being established, despite the popularity of the pneumococcus of Friedländer and Talamon. Though pneumonic fever is to so large an extent a *terra incognita*, there can scarcely be any doubt entertained at the present time in regard to its specific origin. Its remarkable similarity to many of the other specific and self-limited fevers lends powerful support to this view.

I do not believe that true croupous pneumonia can be aborted by any known therapeutic agent, though the severity of many of its symptoms may be greatly abated by judicious treatment. Very early in the disease I employ the opiates and even such heart depressants as aconite and veratrum to allay the pain and to modify the circulation, but I always do so with the greatest caution and only in sthenic cases. The pathological state of the blood, the frequent implication of the pleura and pericardium, the congestion of the bronchial glands, liver, spleen, and other organs, the occasional gastro-intestinal catarrh and cerebral congestion, as

well as the peculiar location of the pneumonic process at the base of the lungs, all seem to me to indicate a poisoned condition of the general circulation. There is a strong probability that a ptomaine or other toxic substance is floating in the blood, and, through its baneful effects upon the nervous system, is the real cause of the disease. As Loomis says, "in local phlegmasiæ there is a direct ratio between the amount of surface involved and the attendant constitutional disturbances." Here, however, there is no sort of a parallelism between the inflammation in its character or its extent and the fever and other symptoms which accompany it. It follows, therefore, that until we find some specific remedy capable of neutralizing this poison in the blood, the treatment of croupous pneumonia must be largely symptomatic.

Those who attempt to formulate a method for all cases alike seem to me to be traveling a wrong road. In the present state of our knowledge, at all events, we must regard each case by itself, study all its individual peculiarities, and treat particular conditions as they arise. Some cases of extreme severity will do well upon one or two remedies persistently administered throughout the entire course of the disease. Others will demand a daily, almost an hourly, modification of the treatment to carry them to a successful issue. And that physician will secure the best results who is the quickest to comprehend the true pathological state at the moment, and who is most completely equipped with the knowledge of a materia medica capable of combating that particular state. Especially should it be remembered that the disease is a specific, self-limited one, that its chief force is manifested in the depression of the nervous system, and that therefore the strength and vitality of the patient must be supported above all other considerations. The case which I have reported demanded a varied treat-

ment, and I believe the fortunate result in one who had so little physical strength previous to the onset of the disease is to be attributed largely to the timely modification of the remedies employed and the free stimulation when the heart began to give out.

My own observation leads me to disapprove of the use of the cold pack and the phenol group of antipyretics in this disease. The nervous depression is far too great to endure the extra depression which these therapeutic agents are liable to add. In fact, I do not believe that the fever is itself so dangerous an element as the nervous exhaustion caused by the noxious influence of the toxic substances in the blood. I am sure a moderate control of the temperature is all that is necessary, and that this control can be obtained to a very large extent by the proper stimulation and nutrition of the patient. As in the other specific self-limited diseases, the maintenance of the vital processes by an abundant selected diet should be the prime object in the treatment. In regard to the choice of stimulants, I usually employ alcohol, nux vomica, and strychnine as my favorites. In some cases digitalis seems to act beneficially and strophanthus more so, but I have learned that it is unwise generally to depend upon them alone. Nux vomica and its alkaloid are both respiratory and cardiac stimulants, and when administered in full dosage as often as the conditions require, I believe they will afford the happiest results.

The above case, as well as many others, establishes to my mind the fact that the heart failure of pneumonia is not due to the high fever and lung obstruction, as contended by many of the systematic writers. These factors may operate in a certain few cases, and to a limited extent may have an influence in all; but there are many impressive facts telling in direct opposition to such a view.

In the first place, the fever of pneumonia is rarely a prolonged high fever. It certainly is not as long or as severe as the fever of many other diseases in which heart failure never occurs. The prolonged high temperature of typhoid, for instance, is so debilitating as to affect the whole organism. The general muscular deterioration of pneumonia is quite marked, and yet the parenchymatous elements of the heart, as shown in post-mortem examinations, do not seem to be degenerated any more than the rest of the system. It is difficult to comprehend how a general pneumonic fever could exercise such a selective action upon the heart if we were to accept the doctrine that the weakness of the heart is due to the fever alone. The heart failure, furthermore, bears no proportionate relation to either the extent or severity of the pulmonic lesion. It is frequently quite as rapid and fatal in cases where the autopsy reveals disease of one lobe as where both lungs are affected.

It is sometimes said that the fever merely weakens the heart, while the extra work imposed upon that organ in forcing the blood through the consolidated lung tissue causes it to collapse. This may be true in some instances, but in very many, of which the case reported is a fair example, the heart does not fail until there is a marked subsidence of the fever and the lung is beginning to clear up. Heart failure may exist before or just when the hepatization of the lung is commencing, and, as Loomis has already pointed out, in many other pulmonary affections there is far greater obstruction to the circulation than there is in pneumonia, and yet they are not accompanied by heart failure. As in the matter of the high temperature, so here it may be argued that there is no proportionate relation between the heart failure and the amount of lung tissue obstructed. In my own case only a comparatively small area of the

lungs was originally affected, and this was beginning to be clear again when the heart broke down. It is difficult to conceive also how the hepatization of the lung should cause so much extra work for the heart, when during the congestive stage of the disease the blood-vessels were overcharged, and yet the action of the heart was then increased rather than diminished. In the consolidation stage of the process the blood-vessels are usually beginning to be relieved of their extra pressure and the circulatory congestion is on the decline. The air cells of the lungs now become the overcrowded elements. Hence, in this stage there is marked percussion dullness and an increased sense of suffocation; but this has all been brought about at the expense of the previously engorged blood-vessels. The blood stasis is compensated, to a certain extent, by the subsequent œdema and transudations. There will be less oxygenation of the blood on account of the diminished supply of air in the lungs, but it is hard to see just how such a state of affairs could produce a mere mechanical obstruction to the flow of the circulation greater, or even as great, as that of the early congestive stage of the disease. If we divide experimentally the cervical portion of the pneumogastric nerve, the lungs undergo a process of hepatization or consolidation which is not associated with heart failure. And yet it is just at the time when hepatization is going on, and not infrequently after it has begun to recede, that the heart ceases work.

Another objection to this mechanical explanation of the heart failure of pneumonia is the suddenness and periodicity with which this unfortunate event takes place. This was illustrated most forcibly in my own case. One would imagine that as the heart muscle grew gradually weaker under the extra strain, and the lungs became more impenetrable under the consolidation process, the failure of the heart

would proceed *pari passu*. But such is not the case. The crisis of the disease occurs more or less regularly at a fixed time and is a comparatively sudden event. In no other disease is there a crisis comparable to that of pneumonia. Other affections have their periods of activity terminated more or less abruptly by death or the beginning of convalescence, when the patient has perhaps reached a state of profound exhaustion, but in pneumonia the crisis is a unique process. All the symptoms continue unabated until a certain period has elapsed, and then in a comparatively sudden, sometimes appalling, manner a complete collapse occurs, with a rapid and remarkable decrease in the temperature and pulse-rate. The picture is not of such a character as to suggest a mechanical explanation; it intimates rather a sudden addition or withdrawal of something abnormal in the circulation. The influence of this something, whatever it may be, is shown pre-eminently through the nervous system. Many think that it is a ptomaine thrown into the blood by the pneumococcus, and that this ptomaine irritates the pneumogastric centers and so causes this nerve to exert an undue inhibitory action upon both the lungs and heart. We know from an examination of the urine that more urea is excreted and that other deleterious substances are retained in the blood that are normally eliminated by the kidneys. As the retention of these poisons in the circulation, however, and the production of the ptomaine by the pneumococcus commence, in all probability, with the commencement of the disease, the question will have to be answered as to why then the heart failure is postponed so long. It is a fact that the heart begins to pulsate irregularly from the very beginning of the disease—so much so that heart insufficiency has been detected by the variations of the pulse within twenty-four hours of the onset of the pneumonia. This is strong presumptive evidence of the

heart trouble being due to the appearance of a particular poison in the blood, but, as I have intimated, it does not satisfactorily explain the sudden heart failure that occurs about the crisis of the disease. It is this latter fact that needs further elucidation for the complete solution of the problem. Furthermore, the explanation is wanting as to the selective action of these poisons upon the pneumogastric nerve and nuclei. If they irritated that nerve, it is quite rational to suppose that they irritate all the nerves of the vaso-motor system. The cardiac ganglia would thus feel the same influence as the respiratory and cardiac centers of the medulla. The motor and inhibitory apparatus would be similarly affected, and thus a kind of compensation would be established which we might look to to prevent the usual suddenness of cardiac failure.

The same brief objections seem to me to apply to the explanation presented by Loomis in Pepper's *System of Medicine* and based upon physiological data. This writer argues that the toxic substance in the blood affects the tonic properties of the vaso-motor system of nerves, so as to allow an undue accumulation of blood within the capillary vessels and arterioles; and as this blood is not returned to the heart, there results a diminution of pressure so great as in itself to cause heart failure. The periodic appearance of the crisis and the comparative abruptness of its phenomena, chief of which is the heart failure, seem to me to have been entirely overlooked in this explanation. Professor Loomis admits that the morbific agent may act upon the intrinsic cardiac ganglia as well as upon the medullary vaso-motor centers. This, as we have said, will doubtless derange the strength and regularity of the heart's action. It will produce, however, a sort of compensation between the augmentation and inhibition of that action.

To sum up, then, I believe that the origin of the whole trouble is a poison floating in the blood and irritating the nerves and nervous centers. The true nature of this poison is as little known as that of many of the other specific constitutional diseases. All explanations of its *modus operandi* must be conjectural until we learn more of its nature. I do not believe that it consists simply of the uneliminated matters of the blood, for in uræmic poisoning and other diseases accompanied by diminished elimination we do not have exactly the same phenomena that we do in the crisis of pneumonia. It is a new substance, and in all likelihood a ptomaine produced by some micro-organism in the lungs. This ptomaine begins to affect the nervous system from the very beginning of the disease, and so causes its constitutional disturbances and general symptomatology. It affects all the nervous elements alike, modifying the regularity of their functions, and, for a time, maintaining a kind of compensation between motor and inhibitory forces. This, of course, exhausts all the tissues involved. About the time of the crisis of the disease, if the process does not extend, the micro-organisms cease to pour their poisonous products into the blood because the cycle of their life history is ended. The influence of these products is thus suddenly withdrawn, and the heart, being therefore deprived of that which, up to the crisis, had been overstimulating and otherwise injuriously affecting it, collapses. This, it seems to me, is, in a few words, the simplest explanation of the heart failure of pneumonia. No one can tell, however, how soon changes may be necessary in all or any explanation as a result of future discovery.

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